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REPORT OF THE UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION*

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I. INTRODUCTION

1. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 1/ presents to the General Assembly, 2/ and thereby to the scientific and world community, its latest evaluations of the sources of ionizing radiation and the effects of exposures. This is the eleventh in a series of reports issued by the Committee since it began its work in 1955. The major aims of the Committee's work are to assess the consequences to human health of a wide range of doses of ionizing radiation and to estimate the dose to people all over the world from natural and man-made radiation sources.

2. The present report and its scientific annexes (see para. 9) 3/ were prepared between the thirty-eighth and the forty-second sessions of the Committee. The material of the report was developed at annual sessions of the Committee, based on working papers prepared by the secretariat that were modified and amended from one session to the next to reflect the Committee's views. The report is based mainly on data provided by Member States until the end of 1989. More recent information has been used in the interpretation of these data.

3. The following members of the Committee served as Chairman, Vice-Chairman and Rapporteur, respectively, at the sessions: thirty-eighth and thirty-ninth sessions: K. Lokan (Australia), J. Maisin (Belgium) and E. Létourneau (Canada); fortieth and forty-first sessions: J. Maisin (Belgium), E. Létourneau (Canada) and L. Pinillos Ashton (Peru); forty-second session: E. Létourneau (Canada), L. Pinillos Ashton (Peru) and G. Bengtsson (Sweden). The names of experts who attended the thirty-eighth to the forty-second sessions of the Committee as members of national delegations are listed in appendix I.

4. In approving the present report, and assuming therefore full responsibility for its content, the Committee wishes to acknowledge the help and advice of a group of consultants appointed by the Secretary-General, who helped in the preparation of the text and scientific annexes. Their names are given in appendix II. They were responsible for the preliminary reviews and evaluation of the technical information received by the Committee or available in the open scientific literature, on which rest the final deliberations of the Committee.

5. The sessions of the Committee held during the period under review were attended by representatives of the United Nations Environment Programme (UNEP), the World Health Organization (WHO), the International Atomic Energy Agency (IAEA), the International Commission on Radiological Protection (ICRP) and the International Commission on Radiation Units and Measurements (ICRU). The Committee wishes to acknowledge their contributions to the discussions.

6. In the present report, the Committee summarizes the main conclusions of the scientific annexes. These results build on previous UNSCEAR reports and take account of the scientific information that has since become available. A major historical review of the Committee's work, including the evolution of concepts and evaluations, was included in the UNSCEAR 1988 report. The present report includes a general introduction to the biological effects of ionizing radiation, based on present understanding (chap. I). In order to quantify the biological effects of radiation and to define the exposures that cause them, it is necessary to understand the radiation quantities and units (see chap. II, sect. A).

7. The consequences of exposures to radiation are assessed (chap. II, sect. B) by making combined use of the results of radiobiological research and the results of epidemiological studies of exposed human populations. The various

sources of human radiation exposures are summarized and evaluated in chapter III. The doses are estimated from information in the published literature, supplemented by data provided by many of the States Members, of the United Nations. Those who make use of the reports of the Committee often have to take account of the way in which people perceive the risks associated with ionizing radiation. These perceptions depend on various personal and societal factors and interactions. The principal features of radiation risk perception are discussed in chapter IV, and a brief summary and some indication of perspectives are given in chapter V.

8. The Committee is aware of the wide readership of the report to the General Assembly and its scientific annexes. Individuals and members of Governments in countries throughout the world are concerned about the possible hazards of radiation. Scientists and medical specialists are interested in the data compilations in the reports of the Committee and in the methodologies presented for radiation assessments. In carrying out its work, the Committee applies its scientific judgement to the material that it reviews and takes care to retain an independent and neutral position in reaching its conclusions. The results of its work are presented for the general reader in the main text of the report to the General Assembly. The supporting scientific annexes are written in a format and a language that are essentially aimed at the specialist.

9. Following established practice, only the main text of the report is submitted to the General Assembly. The full report, including the scientific annexes, will be issued as a United Nations sales publication. This practice is intended to achieve a wider distribution of the findings for the benefit of the international scientific community. The Committee wishes to draw the attention of the Assembly to the fact that the main text of the report is presented separately from its scientific annexes simply for the sake of convenience. It should be understood that the scientific data contained in the annexes are important because they form the basis for the conclusions of the report.

II. BIOLOGICAL EFFECTS OF IONIZING RADIATION

10. The process of ionization changes atoms and molecules. In cells, some of the initial changes may have both short- and long-term consequences. If cellular damage does occur and is not adequately repaired, it may prevent the cell from surviving or reproducing, or it may result in a viable, but modified, cell. The two outcomes have profoundly different implications for the organism as a whole.

11. The function of most organs and tissues of the body is unaffected by the loss of small numbers of cells, or sometimes even of substantial numbers. However, if the number of cells lost in a tissue is large enough and the cells are important enough, there will be observable harm, reflected in a loss of tissue function. The probability of causing such harm is zero at small doses of radiation, but above some level of dose (the threshold) it increases steeply to unity (100 per cent). Above the threshold, the severity of the harm also increases with dose. This type of effect is called deterministic, because it is sure to occur if the dose is large enough. If the loss of cells can be compensated by repopulation, the effect will be relatively short-lived. If the doses are caused by an identified event, it will usually be possible to identify the affected individuals. Some deterministic effects have characteristics that distinguish them from similar effects due to other causes, which may help to identify the affected individuals. The occurrence of an initiating event has sometimes been detected by the unexpected appearance of deterministic effects.

12. The outcome is very different if the irradiated cell is modified rather than killed. It may then be able to produce a clone of modified daughter cells. Within the body there are several highly effective defence mechanisms, but it is not realistic to expect them to be totally effective at all times. Thus the clone of cells produced by a modified but viable somatic cell may cause, after a prolonged and variable delay called the latency period, a malignant condition, a cancer. The probability, but not the severity, of the cancer increases with dose. This kind of effect is called stochastic, which means "of a random or statistical nature". If the damage occurs in a cell whose function is to transmit genetic information to later generations, the effects, which may be of many different kinds and severity, will be expressed in the progeny of the exposed person. That type of stochastic effect is called a hereditary effect. Even if the doses are known, the excess cases of cancer or hereditary disorders can be detected only in a statistical way: the affected individuals cannot be identified. More details are given below.

13. Exposures to radiation are of concern to the Committee mainly in so far as they produce changes in the spectrum of risks to which mankind is subject. It therefore continues to be a major part of the Committee's work to review and interpret data that provide an improved understanding of the quantitative relationships between radiation exposure and effects on health. Except as a result of serious accidents and the unwanted but inevitable irradiation of healthy tissues in radiotherapy, the doses incurred by man are not so large as to produce deterministic effects. Although the Committee continues to take an interest in deterministic effects (one of the annexes to the present report is concerned with deterministic effects in children), most of its biological work in recent years has been concerned with stochastic effects in human beings.

14. The most relevant sources of information on the biological effects of radiation are those obtained directly from studies of human population groups exposed to known and different amounts of radiation. The comparative study of the health of such groups is known as epidemiology. This is a scientific discipline requiring both medical and mathematical skills. It is discussed

further in section I.B. In addition, a great deal of information about the mechanisms of damage and the relationships between dose and the probability of deleterious effects in man can be inferred from biological research on isolated cells grown in vitro and on animals. Studies of this kind allow links to be established between the damage done to cells and the eventual effects in tissues or in the whole organism. It is difficult to make quantitative predictions of the risks to humans from non-human data, but when human data are lacking, animal data may have to be used directly.

15. The main practical interest in the risks of radiation lies in the region of lower doses and dose rates that are experienced in radiation work or in other situations of everyday life. As it happens, however, the strongest epidemiological information comes from situations involving higher doses and dose rates. Some studies at doses of more direct interest, for example, on radiation workers in the nuclear industry and people exposed to radon in houses, are now under way.

16. It is important to realize that epidemiological studies do not have to be based on an understanding of the biological mechanisms of cancer. Their interpretation is greatly improved, however, if they are supported by biological information leading to convincing biological models. These can provide a conceptual basis for interpreting the results of epidemiology, essentially by suggesting dose-response relationships, the parameters of which can be fitted to the observed epidemiological results. The information provided by experimental biology is also supplemented by biophysical knowledge of the initial deposition of energy from radiation in the exposed tissues. The theoretical and experimental results are thus combined to obtain a quantitative relationship between dose and the probability of occurrence of the relevant cancer.

A. Radiobiology

1. The target for radiation action

17. Deoxyribonucleic acid (DNA), the genetic material of the cells, is the most important target for radiation action. There is compelling evidence from in vitro cellular research that the deleterious effects of radiation derive mainly from the damage it causes in cellular DNA.

18. DNA is present in the chromosomes, which are basic components of the cell nucleus. Before every somatic cell division, chromosomes are duplicated so that each daughter cell receives an identical set of chromosomes. Each mammalian species is characterized by a particular and constant chromosome number, size and morphology.

19. In order to explain the mechanisms by which ionizing radiation damages cells, it is necessary to provide a simplified description of the function of the DNA molecule. Although the maintenance of the overall chromosome structure is crucial for several processes involving DNA, it is the DNA polymer itself that is the source of the information that passes from a cell to its descendants. The information is encoded in a linear sequence of alternating molecular structures called base-pairs, which form links between the strands of the double-stranded backbone of the DNA polymer.

20. The base-pair code in DNA is arranged in groups, each providing the basic unit of cellular information and heredity, the gene. In a mammalian cell, it is likely that there are approximately 100,000 genes, each of which depends for its correct function on maintaining a constant base-pair sequence in the DNA.

Changes in those sequences, by base-pair substitution, loss or addition, can change the gene function. Such changes are termed genetic mutations.

21. The DNA is known to be damaged by radiation. Two mechanisms are involved: (a) direct effects of ionization in the DNA structure and (b) indirect effects due to the production of active chemical radicals in the vicinity of the DNA and the diffusion of those radicals to the DNA, where they induce chemical changes. Both direct and indirect effects are of a probabilistic nature, with their probabilities of occurrence increasing with the radiation dose and the volume of the target. There are many other causes of damage to DNA, including errors in replication when cells divide.

22. Damage to the DNA, including radiation damage, is subject to very efficient repair mechanisms mediated by enzyme actions. If the damage to the DNA within a gene is confined to one strand, the repair mechanisms can make use of the information provided by the complementary bases in the other strand. Repair is then highly probable, but, as in any complex system, it is not always error-free. Sometimes, however, both strands may be damaged at the same location in the gene. Repair is then more difficult, and genetic code changes or losses are more likely.

23. A track of radiation consists of a series of separate events, each involving a localized deposition of energy. If this deposition is in the immediate vicinity of DNA and is large enough, molecular damage may occur in the DNA bases or in the backbone strands. The nature and likelihood of the biological damage caused by this DNA damage depends on the density of the energy deposition along the tracks that intersect the DNA and also on the complex interplay between the damage and the repair enzymes of the cell. For sparsely ionizing radiations, such as X-rays, the net effect of these processes is such that the dose-effect relationship for most stochastic effects is curvilinear. Densely ionizing radiations, such as alpha particles and the protons produced by neutrons, are more effective in producing stochastic effects and the dose-effect relationships are more likely to be linear.

24. In addition to these effects at a single point in the DNA, the presence of a number of ion pairs scattered through the nucleus may cause cellular changes that complicate the simple response pattern described above.

25. Irrespective of the detail of the biological mechanism, the probability that radiation will induce specific changes in the genetic code of cells by single tracks and the additional interaction of multiple tracks, may be expressed as the sum of two terms, one proportional to dose and the other proportional to the square of dose. At low doses with any dose rate and at high doses with low dose rate, only the term proportional to dose is effective. At high doses with high dose rate, both terms are relevant. With densely ionizing radiation, for example, alpha particles, there are fewer, but denser, tracks per unit dose, and each track is more likely to produce damage that is not successfully repaired, so the relationship is more likely to be proportional to dose at all doses and dose rates.

26. When human tissues are exposed to radiation, various changes in the cell genetic code (mutations) are induced randomly, with probabilities depending on dose as already discussed. For any given change, the expected number of changed cells is the product of the probability and the number of cells at risk. These cells at risk are considered to be the stem cells of tissues, namely, the cells that maintain the tissues by division, compensating for cells that mature, differentiate and eventually die, in what is called the cell reproduction cycle.

2. Effects of induced changes in the cell genetic code

27. Some changes in the genetic code are incompatible with the sustained reproduction of the cell, resulting in the death of the cell progeny. Unless many cells are killed, this is usually of no consequence for the tissues and organs because of the large number of cells in the tissue and the very substantial redundancy they provide in the functional capability of the tissue.

28. Cell killing by radiation can be quantitatively studied in cell cultures *in vitro* to gain information on the shape of the dose-response relationship. Radiation accidents and experiments *in vivo* with animals show that high doses can deplete the tissues sufficiently to cause functional failure. In turn, deterministic effects in some tissues, such as the vascular and connective tissues, cause secondary damage in other tissues.

29. Other types of change in the genetic code result in viable, but modified, cells. Some of these cells may belong to gonadal cell lines (ova or sperm) and would express the change as hereditary effects. Others would remain in the exposed tissues, being potential causes of somatic effects. In both cases, the effects are stochastic, governed by the probabilistic nature of the induction of changes in the cell genetic code.

(a) Deterministic effects

30. While individual cell killing is a stochastic effect, organ and tissue failures require the killing of large numbers of cells and therefore have thresholds of dose. Cell depletion is a dynamic process operating in competition with the proliferation of unaffected cells. Tissue failures are therefore dependent on both dose and dose rate. Although the changes in individual cells are stochastic, the changes in a large number of cells result in a deterministic outcome. These effects are therefore called deterministic.

31. Because the proportion of cells killed depends on dose, the severity of the deterministic effect also depends on dose. If people of varying susceptibility are exposed to radiation, the threshold in a given tissue for deterministic effects of sufficient severity to be observable will be reached at smaller doses in the more sensitive individuals. As the dose increases, more individuals will incur the observable effect, up to a dose above which the whole group shows the effect.

32. Examples of deterministic effects are the induction of temporary and permanent sterility in the testes and ovaries; depression of the effectiveness of the blood forming system, leading to a decrease in the number of blood cells; skin reddening, desquamation and blistering, possibly leading to a loss of skin surface; induction of opacities in the lens and visual impairment (cataract); and inflammation processes that may occur in any organ. Some effects are indirect in that they are the result of deterministic effects on other tissues. For example, radiation that leads to the inflammation and eventual fibrosis of blood vessels may result in damage to the tissues served by those blood vessels.

33. A special case of deterministic effect is the radiation syndrome resulting from acute, whole-body irradiation. If the dose is high enough, death may result from severe cell depletion and inflammation in one or more vital organs in the body (blood-forming organs, the gastro-intestinal tract and the central nervous system, in decreasing order of sensitivity).

34. During organ development *in utero*, deterministic radiation effects are most pronounced at the time when the relevant tissue is being formed. The killing of even a few, but essential, cells may result in malformations because those cells

will not have progeny. One important effect of exposure to radiation in utero is a dose-related increase in mental impairment, up to and including severe mental retardation.

35. The induction of mental retardation is thought to be the result of the impaired proliferation, differentiation, migration and connection of neural cells at the time when the relevant tissue (brain cortex) is being structured, namely, the 8- to 15-week period after conception in humans. The number of neural cells that are misconnected depends on dose. If, as a first approximation, the magnitude of the mental impairment is taken to be proportional to this number, it would be expected that standard indices of the cognitive functions, for example, the intelligence quotient (IQ), would reflect this dose dependency.

36. In population groups, the IQ has an approximately normal (Gaussian) distribution, conventionally taken to have a central value of 100. Since the average IQ score decreases as radiation dose increases, apparently without an increase in the amplitude of the spread (standard deviation), the decrease in the values of IQ can be described as a uniform shift of the IQ curve to the left (to lower values). If a pathological condition is defined as a condition in which the IQ of an individual is below a stipulated value, such a shift would increase the number of individuals with the pathological condition. This fact is important for the interpretation of the epidemiologically observed mental retardation induced by radiation, which is discussed in section II.B.1.

(b) Cancer induction

37. There is compelling evidence that most, if not all, cancers originate from damage to single cells. Cancer initiation involves a loss of regulation of growth, reproduction and development in somatic stem cells, that is, the loss of control over the cell reproduction cycle and differentiation processes. Point mutations and chromosomal damage play roles in the initiation of neoplasia. Initiation can result from the inactivation of tumour suppressor genes, some of which play a central role in the control of the cell cycle. Although cells may have undergone initiating changes, they will not express their properties until they are stimulated ("promoted") to reproduce by chemicals, hormones etc. in their environment. The promoting agents may be independent of the initiation agent.

38. Single changes in the cell genetic code are usually insufficient to result in a fully transformed cell capable of leading to a cancer; a series of several mutations (perhaps two to seven) is required. In spontaneous cancers, these mutations will have occurred randomly during life. Thus, even after initial cell transformation and promotion, further mutations are needed, and may well be available, to complete the clonal transition from pre-neoplasia to overt cancer. The whole process is called multi-stage carcinogenesis.

39. It is possible that radiation acts at several stages in multi-stage carcinogenesis, but its principal role seems to be in the initial conversion of normal stem cells to an initiated, pre-neoplastic state. The action of radiation is only one of many processes influencing the development of cancer, so the age at which a radiation-induced cancer is expressed is not likely to be very different from that of cancers arising spontaneously. In some circumstances, however, later stages may be affected by radiation, thus changing the times at which cancers appear.

40. Cancer initiation provides the target cells with some degree of proliferative or selective advantage, which is expressed after adequate promotion. The advantage may be a shorter reproduction time than that of normal

cells or a blocking of normal cell differentiation. On the other hand, the very few transformed cells are immersed in a very much larger number of normal cells, and their pre-neoplastic properties can be constrained by their neighbours. An escape from these constraints is a crucial feature of the neoplastic process.

41. Even with their proliferative advantage, transformed cells and their progeny can be eliminated by the random process comprising reproduction, terminal differentiation and death that is at a steady state in mature tissues. The probability of elimination depends on the number of transformed cells and the degree to which they have become autonomous. At least one cell must lead to a clone of modified cells for a cancer to develop. The probability of this occurring is related to dose by the same type of dose relationship (linear or linear-quadratic) as discussed for heritable mutations in the cell. This broadly supports the contention that randomly induced cellular events are responsible for cancer induction.

42. Many animal experiments confirm the predicted shape of the dose-response relationship. It should be mentioned that, at higher doses, cell killing is substantial, competing with cell transformation and causing the dose-response curve to bend downwards. In particular, the following points should be stressed:

(a) Unless the single cell origin of most cancers is thought to be unlikely, no low-dose threshold is to be expected;

(b) If radiation acts primarily as an initiating event, providing one among several required mutations, multiplicative models of risk projection in time can be expected to be more realistic than additive models. (See also sect. II.B.2.)

43. There are problems in assessing the risks of cancer for exposures at low doses and low-dose rates, since most human data are available only at high doses and high-dose rates. The approach commonly used in risk assessment is to fit a linear dose-response relationship to the data, a procedure that is usually considered to give an upper limit to the risk at low doses. This is because the quadratic term will increase the response at high doses with high-dose rates, forcing an increase in the slope of the fitted straight line. From radiobiological considerations, it is then possible to assess the value of the factor by which the slope of the fitted curve should be reduced to give an estimate of the linear component of the linear-quadratic relationship. Direct information on humans exposed at low doses is beginning to emerge and will increasingly provide a check on estimates derived from data at high doses.

44. Novel systems to study cell transformation <u>in vitro</u> and cellular and molecular studies with those systems and with animal neoplasms appear to be potentially very productive sources of information about the mechanisms of cancer induction. Modern cellular and molecular studies may make it possible to differentiate between radiation-induced cancer and other cancers. If samples of tumours from radiation-exposed human groups were to be systematically stored, they would then be a very important resource for future studies on oncogenic mechanisms and for the establishment of causality between cancer in the population and physical or chemical carcinogens in the environment.

(c) <u>Hereditary effects</u>

45. If the change in the genetic code occurs in the germ cells, that is, the egg or sperm or the cells that produce them, the effect is transmitted and may become manifest as hereditary disorders in the descendants of the exposed individuals. Experimental studies on plants and animals show that such changes

may range from trivial to severe, causing gross loss of function, anatomical disorders and premature death.

46. Any non-lethal damage to DNA in germ cells can, in principle, be transmitted to subsequent generations. Hereditary disorders in humans vary widely in their severity. Dominant mutations, that is, changes in the genetic code that produce a clinical effect when inherited from only one parent, can lead to genetic disorders in the first generation progeny. Some of these disorders are very harmful to the affected individual and affect length of life and the likelihood of having offspring. Some dominant mutations can be passed silently through several generations and then suddenly cause their effects. This can occur if the gene is moderated by other genes or is imprinted, that is, if the expression of the gene is dependent on the sex of the parent from whom it was inherited.

47. Recessive mutations are changes in the genetic code that produce a clinical effect only when two copies of the defective gene have been inherited, normally one from each parent. They produce little effect in the first few generations, as most offspring will inherit the defective gene from only one parent, and carriers are usually not affected. However, recessive mutations may accumulate in the gene pool of the population, as each carrier passes the mutation on to many offspring. As the probability that both parents carry the mutation increases, so too does the risk that a child will inherit two copies of the defective gene and will suffer deleterious effects of the mutation.

48. Two points about recessive mutations are important. A recessive mutation often has some effect, albeit slight, even when only a single copy has been inherited, so it may result in some reproductive disadvantage. Also, recessive mutations introduced into the genetic pool are subject to processes that tend to eliminate them: random elimination, called drift, and selection based on reproductive disadvantage. For that reason, newly induced recessive mutations in the genetic pool cause a finite total damage over the generations of descendants.

49. A third, and frequent, type of deleterious change is due to the interaction of several genetic and environmental factors; these are known as multifactorial disorders. A general increase in mutations would be expected to increase the incidence of multifactorial disorders. The magnitude of such an increase is at present unclear but is likely to be small.

B. Epidemiology

50. Epidemiological studies, when interpreted with the aid of biological knowledge, provide the basis for assessing the consequences of radiation exposures. There are also many qualitative studies which confirm that radiation at high enough doses can induce cancer in most of the tissues and organs of the body. There are, however, several significant exceptions. At present, the three principal sources of quantitative information on stochastic effects of radiation in man are the epidemiological studies on the survivors of the nuclear weapon explosions at Hiroshima and Nagasaki, on patients exposed to radiation for diagnostic and therapeutic procedures and on some groups of workers exposed to radiation or radioactive substances at work. As will be seen in this section, there is little hope that differences in exposures to natural sources (excluding radon) will be able to provide quantitative information on stochastic effects, but some occurrences of high radon levels or substantial environmental contamination from accidents may well allow further relevant study groups to be identified.

51. Epidemiology is concerned with establishing patterns in the occurrence of diseases, associating these patterns with likely causes and then quantifying the associations. The process is one of observation and inference. Epidemiological studies are inherently observational in nature: they are arranged by circumstances rather than as a result of experimental design. Choices can be made of the groups to be studied and of the methods of analysing the data, but there is seldom an opportunity to modify the conditions of the study population or the distribution of the causes under investigation. In this way, epidemiology differs sharply from experimental science.

52. Three different types of epidemiological study have been reviewed by the Committee: cohort studies, case-control studies and geographical correlation studies. In cohort studies, a group of individuals, the cohort, is selected on the basis of their exposure to the agent of interest, without prior reference to the disease under study, for example, cancer. The group is then followed forward in time to record the mortality from or the incidence of relevant diseases. The exposure of the members of the cohort to the suspected causative agent is estimated either from contemporary measurements, as in occupational exposure, or by retrospective studies. It is then possible, by standard epidemiological techniques, to compare the incidence of disease or mortality rates following different levels of exposure.

53. If all the members of the cohort have been exposed and there is not a wide enough range of exposures to provide several groups with different levels of exposure, it is necessary to compare the experience of the cohort with that of a control cohort of individuals with substantially lower exposures. Ideally, the two cohorts should be very similar in characteristics that might influence the incidence of or mortality from the disease under study. Otherwise, these characteristics may act as confounding factors, distorting the observed relationship between disease and exposure. Even within a cohort, there may be potentially confounding factors between the groups with different levels of exposure. When information is available on the values of those factors for the individuals in the cohorts, it may be possible to allow for them. The two obvious factors always have to be allowed for in the case of cancer, age and sex. More subtle factors, such as diet, social status and hereditary predisposition, may remain and may be difficult to quantify or even to identify.

54. One important cohort study is the Life Span Study of the survivors of the atomic bombings of Hiroshima and Nagasaki. This is based on a large cohort of all ages and both sexes with a very wide range of exposures. About 60 per cent of the original cohort are still alive, so the present conclusions are still based on incomplete data, especially for those exposed in youth, but it remains the most substantial cohort study used by the Committee.

55. In the second type of study, the case-control study, the aim is to ascertain all the cases of the disease in a defined population, for example, those living in a specified area during a specified period, and then to select for each case one or more control individuals without the disease, but drawn from the same population as the case. The cases and controls can then be compared to see if there are significant differences in the exposures. As with cohort studies, care has to be taken to avoid the effects of confounding factors. This can be done either by matching the controls to the cases for factors such as age and sex or by using statistical techniques in the analysis.

56. Because only the cases and the matched controls have to be investigated, case-control studies can give significant results with smaller study groups than are needed for cohort studies. Case-control studies are therefore useful where the collection of data on the individual exposures requires detailed and extensive fieldwork, making cohort studies impossible or prohibitively

expensive. Case-control studies are particularly useful in examining the effects of exposure to radon in dwellings on the risk of lung cancer. In this work, it is important to allow for smoking habits, for which historical data are usually either lacking or unreliable in cohort studies. The necessary data can be sought in case-control studies.

57. The third type of study is the geographical correlation study. These studies are usually the easiest to conduct, but are the most difficult to interpret and the most prone to error. In a geographical correlation study, two or more groups of people in different locations are selected on the basis of a difference in long-term exposure to radiation, usually radiation from natural sources. Health statistics for the groups are then compared to identify any relevant differences. This technique takes account of the difference in the average exposure between the groups, but ignores the distribution of exposures within the groups, about which information is rarely available. If any important confounding factors, such as age, diet or exposure to pollution, are not randomly distributed between the groups, false conclusions are likely to be reached. Geographical correlation studies have not yet been of much value to the Committee, largely because it is difficult to find groups with a large and accurately known difference in exposure but a small difference in confounding factors.

58. To provide meaningful results, all types of epidemiological study need careful design, execution and interpretation. Moreover, studies that expect a small absolute increase in the incidence of diseases that already exist naturally, such as cancer, must be large if they are to provide statistically significant information. There are two main limitations in epidemiological studies: one, statistical, gives rise to random errors; the other, demographic, gives rise to systematic errors.

59. In many countries, the lifetime probability of dying of cancer is about 20 per cent. If two populations are being compared to detect with confidence the effect of a higher radiation dose in one of them, it is necessary to obtain a difference between them that is statistically significant. To detect an increase in mortality from, say, 20 per cent to 22 per cent, each of the populations would have to number at least 5,000. If the groups were followed to extinction, about 1,000 cancer deaths would be observed in the unexposed group and about 1,100 in the exposed group. The 90 per cent confidence limits on the difference would be about 0-200, just significant. With current estimates of risk, such an increase would result from a lifetime whole-body dose of about 0.4 Sv. This corresponds to an increase by a factor of 5 in the typical lifetime dose from natural sources other than radon (0.001 Sv per year) for the whole 70-year life of the exposed group (0.001 Sv per year × 70 years × 5).

60. The second limitation results from the need to match the study and control groups for any confounding factors that influence the incidence of cancer. Unless the study and control groups are drawn from a single homogeneous population, it is rarely possible to match the groups, or to make allowance for the differences, with sufficient accuracy to detect with confidence a small increase in cancer mortality. Any inadequacy in the matching of the control and study groups may give a bias that cannot be reduced merely by expanding the size of the groups.

61. It is this likelihood of bias that imposes severe limitations on the power of geographical correlation studies of mortality in geographically separated groups such as those used in studies of the effects of exposures to different levels of natural background radiation. It emphasizes the importance of cohort studies, in which a single population can be subdivided into groups with different levels of exposure. There may still be confounding factors that

differ from group to group, but they are likely to be fewer in number than between geographically separated groups. Populations that can be subdivided according to exposure include the Life Span Study group in Hiroshima and Nagasaki, groups of patients undergoing radiotherapy and some occupational groups. Because of those limitations it is important to assess the feasibility of any epidemiological study before committing resources.

62. Much of the quantitative information available from the studies on those populations is limited to fairly high doses and dose rates. Estimates of the risks at smaller doses can be obtained only by extrapolation downwards from the results at high doses. The range of this extrapolation is not large, because the small doses of interest are superimposed on the inescapable doses due to natural radiation sources.

63. In the UNSCEAR 1988 report, the Committee reviewed in detail the high-dose information from epidemiological studies, with an emphasis on the data from Hiroshima and Nagasaki. It is too soon to repeat a comprehensive review of the Japanese data, but it has been possible to take account of the additional data now available and to reassess the previous conclusions. A substantial study of different methods of interpreting the data has been undertaken. In particular, an examination has been made of available models for projecting risk to give estimates of the lifetime probability of death caused by exposure to radiation. The Committee has also made use of other studies, particulary some recently published data on the effects of occupational exposure at moderate to low doses. These data supplement the results from the Life Span Study, but do not yet have the statistical power to add much to the quantitative estimates of risk. The epidemiological studies do not provide significant data for radiation risks in the low-dose range. The extrapolation to the low-dose range has to be validated by experimental biological studies. Therefore the Committee has linked the epidemiological studies with a comprehensive review of the mechanisms of human carcinogenesis and the effects of dose and dose rate on radiation responses. The overall result is to confirm the risk estimates of the 1988 report of the Committee.

64. A great deal of work has been done world wide on epidemiological studies, but the accumulation of quantitative information is necessarily slow. For example, more than half the study group in Hiroshima and Nagasaki is still alive, and the observed excess of cancer deaths, about 350 to date, is rising slowly. The Committee has concentrated its time and resources on extensive scientific discussions on the implications of the available studies and has not prepared an annex on epidemiology for publication at this time. The Committee's conclusions are summarized in section II.B.2 of the present report.

III. QUANTITATIVE ESTIMATES OF RADIATION EFFECTS

A. Quantities and units

65. A specific set of quantities is needed to describe and quantify radiation and its biological effects. Details of radiation quantities and units and an explanation of the derivations and variations in the use of these concepts were presented in the UNSCEAR 1988 report. The Committee's use of quantities and units corresponds to accepted international practice.

1. Dosimetric quantities

66. Radionuclides are characterized by unstable configurations of the nucleus of the atom. They decay in spontaneous nuclear transitions and, in so doing, emit radiation. The characteristic rate of decay of each radionuclide is described by its half-life, the time in which spontaneous transitions will have occurred in one half of the atoms. The rate at which transitions occur in a quantity of a radionuclide is termed the activity, the unit for which is the becquerel (Bq). If a quantity of a radionuclide has an activity of 1 Bq, the transitions are occurring at a rate of one per second.

67. One of the basic quantities used to quantify the interaction of radiation with material is the absorbed dose. This is the energy imparted to a small element of material divided by the mass of that element. The unit of absorbed dose is the joule per kilogram, called for this purpose the gray (Gy). For most purposes, the Committee uses the average absorbed dose in a tissue or whole organism rather than the absorbed dose at a point. Most radiation exposures cause different absorbed doses in different parts of the human body. Absorbed doses from different types of radiation have different biological effectiveness, and the organs and tissues in the body have different sensitivities.

68. For the same absorbed dose, densely ionizing radiations such as alpha particles are more effective in causing biological effects, especially stochastic effects, than are sparsely ionizing radiations such as gamma rays, X-rays or electrons (beta particles). It is useful to combine the absorbed doses from different types of radiation to provide a further quantity called the equivalent dose. The equivalent dose in a human tissue or organ is the absorbed dose weighted by a radiation weighting factor that ranges from unity for sparsely ionizing radiation to 20 for alpha particles.

69. The various organs and tissues in the body differ in their response to exposure to radiation. To allow for this, a further quantity, the effective dose, is used. The equivalent dose in each tissue or organ is multiplied by a tissue weighting factor, and the sum of these products over the whole body is called the effective dose. The effective dose is an indicator of the total detriment due to stochastic effects in the exposed individual and his or her descendants. Since both the radiation weighting factor and the tissue-weighting factor are dimensionless quantities, the dimensions of the equivalent dose and the effective dose are the same as the dimensions of the absorbed dose, and the unit is the same, the joule per kilogram. However, to ensure a clear distinction between the absorbed dose and its weighted analogues, it has been agreed that the unit of equivalent dose and of effective dose should have the special name sievert (Sv).

70. Changes in the radiation and tissue-weighting factors in 1990 complicate the comparisons between new and earlier estimates of dose. In general, the Committee has not attempted to reevaluate old data in terms of the new

quantities, because the changes are usually small. Where reevaluations have been made, this is indicated in the text.

71. Absorbed dose, equivalent dose and effective dose all apply to individuals or to average individuals. The Committee also uses the collective effective dose, which is the average dose to an exposed population or group multiplied by the number of people in the group. This quantity is defined for a specified source or for a specified unit of a practice. It may refer to the total of the future doses committed by that source or unit of practice, as for instance, the collective effective dose committed by atmospheric nuclear explosions or by one year of medical exposures. If the probability of late effects is proportional to effective dose at low doses, which is probably the case, the collective effective dose is an indicator of the total attributable harm to be expected in that group and its descendants. If the individual doses making up a collective dose cover a wide range of values and extend over very long periods of time, it is more informative to subdivide the collective dose into blocks covering more restricted ranges of individual dose and time. The unit of collective effective dose is the man sievert (man Sv).

72. Some events, especially those involving a release of radioactive materials to the environment, may give rise to exposures extending in time, sometimes for many generations. In those situations, the collective dose is still a useful quantity, provided it is made clear that the collective dose is that committed by the relevant source or unit of practice. To give an indication of the dose committed to a typical, but hypothetical, individual now and in the future, the Committee uses the quantity dose commitment. This is the integral over infinite time (or for a specified period) of the average, per caput, dose rate to a specified population, often the world population, resulting from the event. The dose referred to is almost always the effective dose. The dose commitment has been particularly useful in assessing the long-term consequences of events occurring within a limited time, such as a series of atmospheric nuclear explosions. The unit of effective dose commitment is the sievert.

2. Risk and detriment

73. The Committee has also needed to adopt a consistent method of describing quantitatively the probability and severity of stochastic effects of an exposure to radiation. The term risk has been widely used in this context, but without adequate consistency. It is sometimes used to mean the probability of an undesirable outcome, but at other times to mean a combination of the probability and the severity of the outcome. For this reason, the Committee has tried to avoid the use of the term risk, except in well-established formulations such as "excess relative risk" and "multiplicative risk projection model".

74. One important concept for the Committee is the probability of fatal cancer resulting from an increment of exposure to radiation. The annual probability varies with time after exposure, and the most useful summarizing expression is the probability over the whole of life of dying prematurely as the result of the extra exposure. This is not a simple concept, because the total lifetime probability of death is always unity. Any additional exposure to a hazard causing an increase in the probability of death from one cause reduces life expectancy and the probability of death due to all the other causes.

75. For the Committee's purposes, the most appropriate quantity for expressing the lifetime risk of death due to exposure to radiation is the risk of exposure-induced death, sometimes called the lifetime probability of attributable cancer. This quantity takes account of the fact that other causes of death may intervene before the risk of death due to an exposure to radiation can be expressed.

76. Since the effect of the additional exposure is to decrease life expectancy rather than to increase the probability of death, the attributable probability is not an adequate indicator of the effect of an exposure. When summarizing the detriment per unit exposure, the Committee has therefore also used the average period of life lost should an attributable cancer death occur. The combination of this period and the attributable lifetime probability is a measure of the average loss of life expectancy. All these quantities can be used to assess the consequences of a single or continued exposure resulting in a known dose. If the exposures are limited to a range in which the dose-response relationship is approximately linear, the quantities can also be expressed per unit dose. When the relationship is clearly non-linear, the quantities can be specified at a stated dose, usually at an effective dose of 1 Sv.

77. A more complex approach to detriment has been used for protection purposes by the International Commission on Radiological Protection (ICRP). This approach takes account of the attributable probability of fatal cancer in different organs, of the additional detriment from non-fatal cancer and hereditary disorders and of the different latency periods for cancers of different kinds. All these features are included in the selection of the weighting factors for converting equivalent dose into effective dose.

78. The coefficient linking the probability of fatal cancer to the effective dose is thus a function of the age and sex distribution of the exposed population and of any ethnic variations. Nevertheless, the Committee has found it adequate to use the nominal values adopted by ICRP for most of its own purposes, recognizing that these are necessarily approximate, especially in the case of the medical exposure of patients.

B. Effects in man

79. The effects of radiation, outlined in chapter I, section A, can be classified as deterministic or stochastic on the one hand and somatic or hereditary on the other. All deterministic effects are somatic, that is, they occur in the exposed individual, while stochastic effects can be either somatic (for example, radiation-induced cancer) or hereditary.

80. Deterministic effects were quite frequent in the early days of radiation use. During the period between the discovery of X-rays and the early 1930s, when protective measures began to be used, more than a hundred radiologists died of deterministic effects. In addition, there were many cases of anaemia and skin damage. After protective measures were instituted, deterministic effects became progressively less frequent, and they are now seen only in the case of accidents or as a side effect of medical radiation therapy.

81. Cancer induction has been detected and quantified by epidemiology in several exposed groups of people. It appears to be the only stochastic somatic effect of radiation. Hereditary effects of radiation have not yet been epidemiologically identified in humans, but there can be no doubt about their existence. They can be recognized in all the forms of animal and plant life in which they have been sought, other than man. The lack of epidemiological evidence is due to the long time between generations and the large number of people required for statistical detection.

1. Deterministic effects

82. Tissues vary in their deterministic response to radiation. Among the most sensitive tissues are the ovary, the testis, the lens of the eye and the bone

marrow. The threshold for temporary sterility in the male for a single short exposure is about 0.15 Gy, while for prolonged exposures the threshold dose rate is about 0.4 Gy per year. The corresponding values for permanent sterility are in the range 3.5-6 Gy (acute exposures) and 2 Gy per year (chronic exposures). In women, the threshold dose rate for permanent sterility is in the range 2.5-6 Gy for an acute exposure, with women approaching the menopause being more sensitive. For exposures continuing over many years, the threshold dose rate is about 0.2 Gy per year. Those thresholds, like all thresholds for deterministic effects, apply to persons in a normal state of health. For individuals who are already close to exhibiting the effect from other causes, the threshold will be lower. Even in the extreme case where the effect is already present, there will still be a threshold representing the radiation dose needed to produce an observable change in the individual's condition.

83. The threshold for lens opacities sufficient to result, after some delay, in vision impairment is 2-10 Gy for sparsely ionizing radiation (and about 1-2 Gy for densely ionizing radiation) in acute exposures. The threshold dose rate is not well known for long-term chronic exposures, but it is likely to exceed 0.15 Gy per year for sparsely ionizing radiation.

84. For acute exposures of whole bone marrow, the threshold dose for clinically significant depression of blood formation is about 0.5 Gy. The corresponding threshold dose rate for long-term exposure is somewhat above 0.4 Gy per year. Bone-marrow failure is an important component of the radiation syndrome that follows whole-body exposures. An acute whole-body dose of between 3 and 5 Gy causes death in 50 per cent of the exposed population group in the absence of specific medical treatment.

85. In the case of skin exposures, the threshold for erythema and dry desquamation is in the range 3-5 Gy, with symptoms appearing about three weeks after exposure. Moist desquamation occurs after about 20 Gy, with blistering appearing about one month after the exposure. Tissue necrosis, appearing after three weeks, occurs after more than 50 Gy.

(a) Effects on the developing brain

86. Only two conspicuous effects on brain growth and development have emerged from the studies at Hiroshima and Nagasaki. There are some cases of severe mental retardation and some of small head size without apparent mental retardation. Additionally, some groups among those exposed in utero have shown lower than average intelligence scores and poor performance in school.

87. An excess of severe mental retardation was observed in some children exposed to radiation in utero at Hiroshima and Nagasaki. While no mental retardation was observed in cases where exposure occurred before 8 weeks after conception, a sensitive period was identified, 8 to 15 weeks, followed by a substantially less sensitive period of 16 to 25 weeks from conception.

88. As discussed in chapter I, section A.2 (a), the mechanism of mental retardation induction is thought to be the production of a dose-dependent lack of functional connections of neurons in the brain cortex. This lack of connections causes a downward shift (shift to the left) of the IQ distribution, the value of which is estimated to be about 30 IQ points per sievert, for exposures in the period between 8 and 15 weeks.

89. Normal IQ distributions have a stipulated average value of 100 IQ points and a standard deviation of about 15 IQ points. The region to the left of two standard deviations from the average, that is, values less than 70 IQ points, corresponds to the clinical designation of severe mental retardation. The

radiation-induced shift, for a dose of 1 Sv, would result in severe mental retardation in about 40 per cent of the exposed individuals.

90. Bearing in mind the shape of the Gaussian distribution, however, the fraction of extra cases caused by the shift induced by a small dose would be substantially less than that calculated directly from a linear relationship of 40 per cent per sievert (about one order of magnitude less). The dose required to cause an IQ shift large enough to make an otherwise normal individual severely mentally retarded would be high (in the region of 1 Sv or more), while the dose required to bring an individual, who without radiation exposure would have a low IQ, into the category of severely retarded, by crossing the borderline, might be a few tenths of a sievert.

(b) Effects in children

91. During childhood, when tissues are actively growing, radiation-induced deterministic effects will often have a more severe impact than they would during adulthood. Examples of deterministic damage resulting from radiation exposure in childhood include effects on growth and development, organ dysfunction, hormonal deficiencies and their sequelae and effects on cognitive functions. Most of the information comes from patients who have received radiotherapy and is derived by new analytical methods and by continued careful monitoring. The Committee has reviewed this information to identify the nature of the effects in various tissues and the magnitude of the doses causing these effects.

92. Many factors complicate the study of the dose-effect relationship. These include the underlying disease and the modality of the treatment, which often includes surgery and chemotherapy in addition to the radiotherapy. For those reasons, the estimates of threshold doses in healthy children are still qualified by substantial uncertainties. Only general indications of levels can be provided. Unless otherwise stated, the doses are from fractionated exposures.

93. The effects of radiation on the testis and the ovary are dependent on both age and dose. Testicular function can be compromised at doses of 0.5 Gy. At doses of 10 Gy, gonadal failure occurs in most irradiated boys. In girls, a small proportion show amenorrhea following doses of 0.5 Gy, the proportion increasing to about 70 per cent at doses of 3 Gy. Infertility occurs in about 30 per cent of cases following doses of 4 Gy. A dose of 20 Gy results in permanent infertility in all cases.

94. Many other organs are damaged by doses in the range 10-20 Gy. In contrast, thyroid damage may occur at doses as low as about 1 Gy. Several effects have been shown in the brain, including atrophy of the cortex, after a single dose of 10 Gy or an accumulated dose of 18 Gy delivered in about 10 fractions. The endocrine system is affected by radiation, showing clearly impaired secretion of growth hormones at fractionated doses totalling 18 Gy. Thyroid doses in the region of 1 Gy, protracted over two weeks, resulted in hypothyroidism in patients treated by cranial radiotherapy. Cataracts and impairment of breast development have been seen at 2 Gy.

95. Deterministic effects in several other organs have been identified and quantified. Reduced total lung capacity has been shown at doses of 8 Gy and restrictive lung changes at doses of 11 Gy. Five exposures per week over six weeks require a total dose of more than 12 Gy to produce liver damage, and protracted doses of about 12 Gy are sufficient to produce kidney damage. Radiation nephritis has been reported at 14 Gy. A dose exceeding 20 Gy is required to stop bone formation, with partial effects following doses in the

range 10-20 Gy and no effects below 10 Gy. Damage to the heart muscle leading to clinical failure is seen after a dose of about 40 Gy.

2. Radiation-induced cancer

96. Mechanistic models for the induction of cancer by radiation can be formulated from radiobiological information: these models suggest the choice of the dose-response function. Human epidemiology provides the data to be interpreted using such models, which are particularly important in the extrapolation of the data to the low-dose region, where epidemiological data are lacking or are extremely imprecise.

97. Since the period of observation of an exposed population sample rarely extends to a full lifetime, it is usually necessary to project the frequency of cancer induction noted during the period of observation to the lifetime of the exposed population, in order to obtain the full lifetime risk. Two principal models have been used for this purpose, one the absolute, or additive, projection model and the other the relative, or multiplicative, model.

98. The simple absolute (additive) model assumes a constant (dose-related) excess of induced cancer throughout life, unrelated to the age-dependent spontaneous rate of cancer. The simple relative (multiplicative) model, assumes that the rate of induced cancers will increase with age as a constant multiple (dose-related) of the spontaneous cancer rate. Both models may be extended to replace the constant values by functions of age at exposure and of time since exposure.

99. The simple additive model is no longer seen to be consistent with most epidemiological observations, and radiobiological information seems to favour the multiplicative model. It should be noted, however, that neither of the simple models fits all the information; for example, the multiplicative model has difficulties with the case of exposure of young children, and neither of the simple projection models is consistent with the data for leukaemia or bone cancer.

100. Three projection models for solid cancers have been examined by the Committee. The first is the simple model with a constant excess risk factor. The second and third use a decreasing factor for times more than 45 years after exposure. Although the leukaemia risk is not yet fully expressed in the Japanese survivors, the residual risk is now sufficiently small to make the use of different projection models unnecessary.

101. The two models with decreasing relative risk factors reduce the estimates of lifetime risk following a single exposure by a factor of about 2 for exposure in the first decade of life and by a factor of 1.5 in the second decade, with only a small effect for older ages at exposure. Because the reduction in probability occurs at older ages, these models show slightly larger loss of life per attributable cancer than does the simple model.

102. An important element in the assessment of the radiation risks of cancer at low doses is the reduction factor used to modify the direct linear (non-threshold) fit to the high-dose and high-dose-rate epidemiological data in order to estimate the slope of the linear component of the linear-quadratic function. From basic radiobiological information, animal studies, and data relevant to cancer induction in man, this factor is now estimated, with substantial uncertainty, to be about 2 for the dose range providing most of the epidemiological data. The epidemiology results do not exclude this value, but, except for leukaemia, they do not support it.

103. In the UNSCEAR 1988 report, the Committee derived risk coefficients (risk per unit dose) for high-dose and high-dose-rate situations for various tissues. For the purpose of this report, it is sufficient to deal with the total risk of cancer mortality when the whole body is exposed.

104. In recent years, epidemiological studies have been reported on occupationally exposed persons, on population groups living in areas having different levels of background radiation and on people exposed by the release of radioactive materials to their environment. For such studies to provide useful quantitative information on the consequences of exposure to radiation, they must be of a substantial size and must be extended over long periods. Historically, only the studies of radon-related lung cancer in miners have been able to provide quantitative relationships, and these are specific to radon. At present, the most promising studies of general application are those of workers exposed to several kinds of radiation in the course of their work. These studies are now beginning to show positive results.

105. The statistical power of these studies is still low, but it will increase with time as the data accumulate. The results are consistent with those from studies at high doses and high dose rates and provide no indication that the current assessments underestimate the risks.

106. The data now indicate with reasonable certainty that the cancer risks associated with high doses of sparsely ionizing radiation are about three times greater than they were estimated to be a decade ago. The 1988 estimate of probability of lifetime fatal cancers using the preferred multiplicative risk projection model was 11 10^{-2} per Sv for the exposed populations at Hiroshima and Nagasaki, of whom more than half in the epidemiological study are still alive. The Committee's estimates relate only to the Japanese population represented by the Life Span Study cohort. These studies are continuing, but there is as yet insufficient information to suggest a change in the risk estimates.

107. The Committee discussed the factor by which risk estimates derived from studies at high doses should be reduced when used to derive estimates for low doses. No single figure can be quoted, but is clear that the factor is small. The data from the Japanese studies suggest a value not exceeding 2. If a factor of 2 is used, a value of 5 10^{-2} per Sv is obtained for the lifetime probability of radiation-induced fatal cancers in a nominal population of all ages. A smaller average value of about 4 10^{-2} per Sv would be obtained for a working population (aged between 18 and 64 years) exposed during their working lives. The Committee suggests that a reduction factor should be applied for all doses below 0.2 Gy and for higher doses when the dose rate is less than 6 mGy per hour averaged over a few hours.

3. Hereditary effects

108. Epidemiology has not detected hereditary effects of radiation in humans with a statistically significant degree of confidence. The risk estimate based on animals is so small that it would have been surprising to find a statistically significant effect in the end-points studied in Hiroshima and Nagasaki. Nevertheless, there can be no doubt of the existence of hereditary effects in man. Risk estimation therefore rests on genetic experimentation with a wide range of organisms and on cellular studies, with limited support from the negative human findings.

109. Two considerably different methods of estimating genetic risk have been used by the Committee. One is the doubling dose (or indirect) method. This assessment excluded the multifactorial disorders. For a reproductive

population, a risk value of 1.2 10^{-2} per Sv was given for all generations after exposure or, expressing the same risk in a different way, a risk of 1.2 10^{-2} per generation for a continued exposure of 1 Sv per generation. The corresponding risk in the first two generations after exposure was estimated to be 0.3 10^{-2} per Sv in the reproductive segment of the population.

110. The Committee's other method of assessing genetic risk is the so-called direct method. It applies to clinically important disorders expressed in first-generation offspring of exposed parents. The estimate of risk was 0.2-0.4 10^{-2} per Sv in the reproductive part of the population. It is reassuring that the two different methods of genetic risk assessment give reasonably similar estimates.

111. There are many diseases and disorders of complex, multifactorial aetiology. In addition, there are a number of newly recognized, non-traditional, mechanisms of transmitting hereditary disease. The effect of radiation upon the incidence of these multifactorial and non-traditionally transmitted diseases is highly speculative, but may be slight. More research is needed to make it possible to derive risk estimates for all of the mechanisms that could cause diseases in the offspring of exposed individuals.

IV. SOURCES OF RADIATION EXPOSURE

A. Basis for comparisons

112. The radiation to which the human population is exposed comes from very diverse sources. While some of these sources are natural features of the environment, others are the result of human activities. The radiation from natural sources includes cosmic radiation, external radiation from radionuclides in the earth's crust and internal radiation from radionuclides inhaled or ingested and retained in the body. The magnitude of these natural exposures depends on geographical location and on some human activities. Height above sealevel affects the dose rate from cosmic radiation; radiation from the ground depends on the local geology; and the dose from radon, which seeps from the ground into houses, depends on local geology and on the construction and ventilation of houses. The exposures due to cosmic rays, terrestrial gamma rays and ingestion vary only slightly with time, so they can be regarded as the basic background exposure to natural sources.

113. Man-made sources of radiation include X-ray equipment, particle accelerators and nuclear reactors used in the generation of nuclear energy, in research and in the production of radionuclides that are then used in medicine, research and industrial operations. Past testing in the atmosphere of nuclear devices still contributes to world-wide exposures. Occupational exposure, that is, the exposure of workers, is widespread, but involves groups of limited size.

114. Some sources of exposure, for example, natural sources, can be viewed as continuing at a constant level. Others, for example, medical examinations and treatments and the generation of nuclear power, continue over long periods, not necessarily at a constant level. Still others, for example, test explosions in the atmosphere and accidents, are discrete events or a discrete series of events. Sources that release radioactive materials to the environment deliver their doses over prolonged periods, so that the resulting annual doses do not provide a satisfactory measure of their total impact.

115. Given those complexities, there is no satisfactory single way of presenting the resultant dose to man. However, there is some advantage in attempting a compromise presentation that allows all the sources to be seen on a common basis, while preserving a more selective presentation for the details of the exposure from each type of source. One method is to present the average annual doses from various sources up to the present time. This type of presentation demonstrates the historical significance of the sources to date, but gives no indication of any future dose already committed. The Committee has partially avoided that difficulty by using the dose commitment, which takes account of future doses committed by the source. However, neither the dose commitment to date nor the collective dose committed to date provides an adequate representation of the doses from practices that are likely to be continued into the future. For this, some system of forecasting is needed.

116. The approach to be used in this report to compare radiation exposures from various sources consists of presenting the collective dose to the world population received or committed (a) from the end of 1945 to the end of 1992 (47 years) for discrete events and (b) for a period of 50 years at the current rate of practice or exposure for all other sources, including natural sources. This approach assumes that the current rate of practice is reasonably typical of a period of 50 years, 25 years before and after the present. It is likely that this assumption overestimates the future doses from practices that are not rapidly expanding, because improved techniques and standards of protection will

reduce the doses per unit of practice. No assumption is needed for discrete events.

117. This chapter summarizes the Committee's evaluation of exposures of the public and workers to radiation from the various sources. The detailed information is to be found in the scientific annexes to the present report.

B. Levels of exposure

1. Exposures from natural sources

118. The world-wide average annual effective dose from natural sources is estimated to be 2.4 mSv, of which about 1.1 mSv is due to the basic background radiation and 1.3 mSv is due to exposure to radon. The cosmic ray dose rate depends on height above sealevel and on latitude: annual doses in areas of high exposure (locations at the higher elevations) are about five times the average. The terrestrial gamma-ray dose rate depends on local geology, with a high level typically being about 10 times the average. The dose to a few communities living near some types of mineral sand may be up to about 100 times the average. The dose from radon decay products depends on local geology and housing construction and use, with the dose in some regions being about 10 times the average. Local geology and the type and ventilation of some houses may combine to give dose rates from radon decay products of several hundred times the average.

119. Table 1 shows typical average annual effective doses in adults from the principal natural sources. With the accumulation of further data and minor changes in the methods of assessment, the estimate of the annual total has been almost constant: 2.0 mSv in the UNSCEAR 1982 report, 2.4 mSv in the UNSCEAR 1988 report and 2.4 mSv in table 1.

120. The typical annual effective dose of 2.4 mSv from natural sources results in an annual collective dose to the world population of 5.3 billion people of about 13 million man Sv.

Table 1. Annual effective doses to adults from natural sources

Source of exposure	Annual effective dose (mSv)	
	Typical	Elevated*
Cosmic rays	0.39	2.0
Terrestrial gamma rays	0.46	4.3
Radionuclides in the body (except radon)	0.23	0.6
Radon and its decay products	1.3	10
Total (rounded)	2.4	-

* The elevated values are representative of large regions. Even higher values occur locally.

2. Medical exposures

121. Wide use is made of radiation in diagnostic examinations and in treatments. Of these, diagnosis is by far the more common. Most people are familiar with X-ray examinations of the chest, back, extremities and gastro-intestinal tract and dental X-rays, as these are the examinations most frequently performed. The

provision of medical radiation services is, however, very uneven in the world, with most of the procedures being carried out in industrialized countries, which contain only one quarter of the world's population.

122. Based on a correlation between the numbers of medical X-ray equipment and examinations and the number of physicians in countries, the Committee has evaluated medical radiation exposures for four levels of health care in the world, from level I in industrialized countries to level IV in the least developed countries. This broad classification is useful, but it sometimes conceals substantial variations within countries.

123. As health care improves, countries move between health-care levels. Thus, the number of people living in the different categories of countries changes with time. Between 1977 and 1990, the greatest change was an increase of population in level II countries from about 1.5 billion to about 2.6 billion. The estimates for 1990 show level I at 1.35 billion, level II at 2.63 billion, level III at 0.85 billion, and level IV at 0.46 billion.

124. Representative estimates of examination frequencies and doses per examination have been obtained from a world-wide survey conducted by the Committee. For countries of health-care level I, the annual frequency of medical (that is, non-dental) X-ray examinations was 890 per 1,000 population. For levels II, III and IV, the frequencies per 1,000 were 120, 70 and 9. The number of examinations is closely proportional to the number of physicians. In each level, there are differences within and between countries, with most countries lying within a factor of about 3 from the mean of the health-care level. The spread is wider in countries at the lower health-care levels.

125. The doses per examination are generally low, but there is a wide range both within and between countries. The data from level II, and more particularly from levels III and IV, are very limited, but show no obvious differences from level I data. Despite the low doses per examination, the magnitude of the practice makes the diagnostic use of X-rays the dominant source of medical radiation exposures. Nevertheless, doses from the use of radiopharmaceuticals and from therapeutic treatments have also been evaluated.

126. Patient doses are expressed in terms of effective dose. This permits comparisons between time periods, countries, health-care levels, medical procedures and sources of exposure. However, patients differ from the population at large in age- and sex-distribution and in life expectancy, so the nominal fatality coefficients discussed in chapter II, section A, are only very approximate.

127. When considering the implications of the dose to patients, it is important not to lose sight of the associated benefits. Reducing an individual dose in diagnosis will decrease the detriment to the patient, but it may also decrease the amount or quality of the diagnostic information. In therapy, too small a dose may completely eliminate the benefit of the treatment. In screening studies, the benefit of early detection of a condition must take account of the consequent opportunity for improved management of the individual case, because detection alone is not necessarily beneficial. Collective dose can be a misleading basis on which to make judgements. In many countries, an increase in collective dose would signal an increase in the availability of health care and a net increase in benefit.

128. Information on the mean annual effective dose per patient from X-ray diagnosis is available from 26 countries, of which 21 were in level I, 4 in level II, and 1 in level III. In countries of level I, there has been a widespread downward trend in the dose per patient for most types of examination.

The notable exception is in computed tomography, where the doses have tended to increase. In the countries for which data are available, the values of the annual effective dose per patient are mainly within the range 0.5-2.0 mSv. For individual examinations, values may fall outside this range, being lower for examinations of the extremities and skull and higher for examinations of the gastro-intestinal tract.

129. The annual effective dose per caput is available from 21 countries in level I, 5 in level II, and 2 in level III. The values in level I show a range of 0.3-2.2 mSv. It is not easy to make reliable estimates for countries in the lower levels of health care. For levels II and III, however, the range seems to be about 0.02-0.2 mSv. The population-weighted average for level I is 1.0 mSv, the same as reported in 1988. The average for the world is 0.3 mSv. One cause of uncertainty in these values is the use of fluoroscopy. This procedure results in much higher doses than those from radiography, and its prevalence is both uncertain and changing with time.

130. The diagnostic use of radiopharmaceuticals has stabilized in countries of level I, but is probably increasing in countries of levels II-IV. There have been significant changes of technique in this field. The use of long-lived nuclides in developing countries results in a higher dose per examination than in countries where short-lived alternatives are available. In particular, the use of iodine-131 has decreased sharply, although it still contributes substantially to the collective dose in industrialized countries. The annual effective dose per caput is still only about 10 per cent of that attributable to the diagnostic use of X-rays. For countries of level I, the annual effective dose per caput is about 0.09 mSv. For countries of lower health-care levels, it is an order of magnitude less. World wide, the annual effective dose per caput from diagnostic nuclear medicine is 0.03 mSv.

131. The estimated annual effective dose per caput from all diagnostic uses of radiation is 1.1 mSv in countries of health-care level I and about 0.3 mSv averaged over the whole world. The annual collective effective dose world wide from diagnostic medical exposures is about $1.8\ 10^6$ man Sv. This is the largest exposure from man-made sources or practices and is equal to about one seventh of the annual collective dose to the world's population from natural sources of radiation.

132. The dose to individual patients undergoing radiotherapy is very much higher than in diagnosis, but the number of patients is smaller. There are difficulties in defining an appropriate quantity for expressing dose outside the target organ. The Committee has used a quantity analogous to effective dose, but ignoring the dose to the target tissue. For most practical purposes, this quantity may be considered the same as the effective dose.

133. With this simplification, the world-wide annual total collective effective dose from therapy is about $1.5\ 10^6$ man Sv, about the same as that from diagnosis. The comparison of doses in diagnosis and therapy may not, however, correctly reflect the relative detriment. The difference in age distributions does not appear to be marked, but the subsequent expectation of life is likely to be less for the therapy patients. This gives less time for late effects to develop and thus reduces the relative detriment.

134. Exposures from medical radiation usage can be expected to increase as populations age and become urbanized and as health-care services spread throughout the world. There are also, however, trends towards lower doses per examination and the substitution of alternative techniques, such as imaging by magnetic resonance and ultrasound. There will be great differences in the trends in countries of different levels of health care.

3. Exposures from nuclear explosions and from the production of nuclear weapons

135. Nuclear explosions in the atmosphere were carried out at several locations, mostly in the northern hemisphere, between 1945 and 1980. The periods of most active testing were 1952-1958 and 1961-1962. In all, 520 tests were carried out, with a total fission and fusion yield of 545 Mt.

136. Since the Treaty Banning Nuclear Weapon Tests in the Atmosphere, in Outer Space and Under Water, was signed at Moscow on 5 August 1963, almost all nuclear test explosions have been conducted underground. Some of the gaseous fission products were unintentionally vented during a few underground tests, but the available data are insufficient to allow an assessment of the resultant dose commitment. The total explosive yield of the underground tests is estimated to have been 90 Mt, much smaller than that of the earlier atmospheric tests. Furthermore, although the underground debris remains a potential source of human exposure, mainly locally, most of it will be contained. The earlier atmospheric tests therefore remain the principal source of world-wide exposure due to weapons testing.

137. The total collective effective dose committed by weapons testing to date is about $3\ 10^7$ man Sv. Of this, about $7\ 10^6$ man Sv will have been delivered by the year 2200. The rest, due to the long-lived carbon-14, will be delivered over the next 10,000 years or so. Another way of expressing these findings is to use the integral over time of the average dose rate to the world population, the dose commitment. The dose commitment to the year 2200 from atmospheric testing is about 1.4 mSv; over all time, it is 3.7 mSv. Both figures are of the same order of magnitude as the effective dose from a single year of exposure to natural sources. The fraction of the dose commitment delivered by 2200 (38 per cent) is not the same as the fraction of the corresponding collective dose (23 per cent) because the world population is expected to rise from 3.2 billion at the time of the main weapon testing programmes to a constant 10 billion for most of the 10,000 years.

138. These global estimates include a contribution from the doses to people close to the sites used for atmospheric tests. Although that contribution is small in global terms, some local doses have been substantial. The thyroid doses to children near the Nevada test site in the United States may have been as much as 1 Gy. Similar, but somewhat larger, thyroid doses were incurred between 1949 and 1962 in settlements bordering the Semipalatinsk test site in the former USSR. Some doses near the Pacific test site in the United States were also high, largely because the wind changed direction after one thermonuclear test. Ground contamination near Maralinga, Australia, the site of British nuclear tests, has been sufficient to restrict subsequent access. Without further decontamination, unrestricted continuous occupancy might cause annual effective doses of several millisieverts in two areas, with values up to 500 mSv in small areas immediately adjacent to the test sites. The local and regional collective effective dose from the whole test series was about 700 man Sv.

139. The operations needed to produce the world supply of nuclear weapons are also a source of exposure. The processes start with the mining and milling of uranium. The uranium is then enriched, either to a high degree for weapon components or slightly for use in reactors producing plutonium and tritium. The scale of those activities is not publicly available and has to be assessed indirectly. The resultant dose commitments are then estimated by applying dose per unit release factors from nuclear power production, for which more data are freely available. The local and regional collective effective dose to the public committed by these operations is estimated to be about 1,000 man Sv. The

global collective dose will be larger by a factor of between 10 and 100. Even if the total collective dose is taken to be 10^5 man Sv, it is a small fraction of the collective effective dose committed by the test programmes.

140. As in the case of testing, some local doses have been substantial. The doses near the plutonium production plant at Hanford, Washington, United States, are currently being evaluated. Preliminary results suggest that thyroid doses might have been as high as 10 Gy in some years in the 1940s. The release to the environment of the wastes from the processing of irradiated fuel at the Soviet military plant near Kyshtym, in the Ural mountains, resulted in cumulative effective doses of about 1 Sv at some riverside locations up to 30 km from the site over a few years in the early 1950s.

4. Exposures from nuclear power production

141. The generation of electrical energy in nuclear power stations has continued to increase since the beginning of the practice in the 1950s, although now the rate of increase is less than that for electrical energy generation by other means. In 1989, the electrical energy generated by nuclear reactors was 212 GW a, 17 per cent of the world's electrical energy generated in that year. The total electrical energy generated by reactors from the 1950s until 1990 was slightly less than 2,000 GW a.

142. As in previous UNSCEAR reports, the collective effective dose committed by the generation of 1 GW a of electrical energy by nuclear sources has been estimated for the whole of the fuel cycle from mining and milling, through enrichment, fuel fabrication and reactor operation, to fuel reprocessing and waste disposal. No specific allowance has yet been made for decommissioning, partly because of the limited experience available to date and partly because it is already clear that the contribution is likely to be small.

143. Detailed information was obtained on the releases of radionuclides to the environment during routine operations from most of the major nuclear power installations in the world. From that information, the Committee has assessed normalized releases per unit of electrical energy generated. The collective effective doses committed per unit energy generated were then estimated with the help of the generalized environmental models developed by the Committee in previous UNSCEAR reports. Separate estimates were made for the normalized components resulting from local and regional exposures and from exposures to globally dispersed radionuclides. The main contributions are shown in table 2. These committed collective doses were truncated at 10,000 years because of the great uncertainties in making predictions over longer periods.

144. The value of 3 man Sv $(GW\ a)^{-1}$ for the normalized local and regional collective dose committed per unit of energy generated is slightly smaller than the value estimated in previous reports. The main reductions have been in reactor operation and reprocessing, with some increase in the estimates for mining and milling. The current value is therefore not representative of the entire period of nuclear power production, the normalized dose in the earlier part of the period being somewhat higher than the average. The total collective dose committed by effluents released from the nuclear fuel cycle up to the end of 1989 is estimated to be slightly more than 10,000 man Sv. The collective dose committed by globally dispersed radionuclides and by solid waste disposal is uncertain, since it depends on future waste management practices and the

Table 2. Normalized collective doses to the public from nuclear power production

Source	Collective effective dose committed per unit energy generated [man Sv $(GW\ a)^{-1}$]
Local and regional component	
Mining, milling, and tailings	1.5
Fuel fabrication	0.003
Reactor operation	1.3
Reprocessing	0.25
Transportation	0.1
Total (rounded)	3
Global component (including solid waste disposal)	
Mine and mill tailings (releases over 10,000 years)	150
Reactor operation waste disposal	0.5
Globally dispersed radionuclides mainly from reprocessing and solid waste disposal	50
Total (rounded)	200

evolution of the world's population over the next 10,000 years. Using the estimate of 200 man Sv $(GW\ a)^{-1}$ shown in table 2, the total nuclear power generated, 2,000 GW a, is estimated to have committed a collective effective dose of 400,000 man Sv.

145. If the current rate of generation and the normalized values of table 2 are representative of the 50-year period centred on the present, the 50-year collective effective dose from nuclear power generation is about 2 106 man Sv.

146. The doses to individuals from the generation of electrical energy differ very widely, even for people near similar plants. Some estimates of the maximum doses have been made for realistic model sites. For the principal types of power plants, the annual effective doses to the most highly exposed members of the public range from 1 to 20 μSv. The corresponding annual figures for large fuel reprocessing plants are 200-500 μSv.

5. Exposures of the public from major accidents

147. As in all human activities, there are accidents at work. The exposure of patients to radiation for diagnostic or therapeutic reasons is also subject to failures of equipment or procedures. The doses resulting from minor mishaps at work are included in the routine monitoring results. Some accidents, both occupational and medical, have serious consequences for the individuals involved. Such accidents are fairly frequent (perhaps a few hundred each year world wide), but the probability that any given member of the public will be involved is very small. The present section deals only with the major accidents affecting members of the public.

148. The production and subsequent transport of nuclear weapons have resulted in several accidents. The transport accidents caused local contamination by plutonium. The collective dose committed by those accidents is small. In one accident, at Palomares, Spain, the highest committed effective dose was about

200 mSv. Other accidents on land and the loss of nuclear weapons at sea have caused negligible doses to people.

149. The two most serious accidents in nuclear weapons production were at Kyshtym in the southern Ural mountains of the Soviet Union in September 1957 and at the Windscale plant at Sellafield in the United Kingdom in October of the same year.

150. The Kyshtym accident was a chemical explosion following a failure of the cooling system in a storage tank of high-activity waste fission products. The principal fission products released were isotopes of cerium, zirconium, niobium and strontium. The doses were due to fission products deposited on the ground and strontium entering the food chain. The collective dose was shared about equally between those who were evacuated from the area of high contamination (about 10,000 people) and those who remained in the less contaminated areas (about 260,000 people). The total collective dose over 30 years was estimated to be about 2,500 man Sv. The highest individual doses were to people evacuated within a few days of the accident. The average effective dose for this group of 1,150 people was about 500 mSv.

151. The Windscale accident was a fire in the natural uranium and graphite core of an air-cooled reactor primarily intended for the production of military plutonium. The principal materials released were isotopes of xenon, iodine, caesium and polonium. The most important route of intake was the ingestion of milk, which was controlled in the area near the accident. Further away, the uncontrolled consumption of milk and inhalation were significant sources of exposure, with iodine-131 and polonium-210 being the two most important nuclides. The total collective effective dose in Europe, including the United Kingdom, was about 2,000 man Sv. The highest individual doses were to the thyroids of children living near the site. These ranged up to about 100 mGy.

152. There have been several accidents that have damaged nuclear power reactors, of which the accident at Three Mile Island in the United States and Chernobyl in the Soviet Union were the most important. The Three Mile Island accident caused serious damage to the core of the reactor, but almost all the fission products were retained by the containment system. The resulting collective effective dose was not more than about 40 man Sv. The doses to individual members of the public were low, the highest dose having been slightly less than 1 mSv.

153. The Chernobyl accident was discussed in detail in the UNSCEAR 1988 report (sect. III.A.8). The explosion and subsequent graphite fire released a substantial fraction of the core inventory and caused a distribution of effective doses in the northern hemisphere, mainly in the Soviet Union and Europe. The collective effective dose committed by the accident is estimated to have been about 600,000 man Sv. The doses to individuals varied widely, with a few people in the evacuated group receiving effective doses approaching 0.5 Sv. The average annual effective dose in the strict control zones surrounding the evacuation area fell from about 40 Msv in the year following the accident to less than 10 mSv in each of the years up to 1989.

154. An international review of the situation in the zones around the evacuation area was conducted in 1990. The project corroborated the estimated doses and found that the health of the population at that time was comparable to that of the population in nearby uncontaminated settlements.

155. Sealed sources used for industrial or medical purposes are occasionally lost or damaged and members of the public injured. Four severe accidents of this kind have occurred since 1982. In Mexico, in 1983, an unlicensed teletherapy source containing cobalt-60 was sold as scrap metal. Apart from the

widespread contamination of steel products in Mexico and the United States, about 1,000 people were exposed to substantial levels of radiation, with effective doses up to about 250 mSv. About 80 people received higher doses, up to 3 Sv, and seven received doses in the range 3-7 Sv. There were no deaths.

156. In Morocco, in 1984, eight members of one family died after they found and kept at home a sealed industrial radiography source containing iridium-192. The effective doses were in the range 8-25 Sv. In Goiania, Brazil, in 1987, a caesium-137 teletherapy source was removed from its housing and broken up. Severe doses were received from direct radiation and from the localized contamination. Doses to individuals ranged up to 5 Sv. Fifty-four people were hospitalized and four died. In Shanxi Province, China, in 1992, a cobalt-60 source was lost and picked up by a man. Three persons in the family died of overexposure. In 1993, an accident occurred at a plant near Tomsk in the Russian Federation. The information on this accident has not yet been fully assessed, but it appears that the exposures were very low and that few members of the public were involved.

6. Occupational exposures

157. Occupational radiation exposures are incurred by several categories of workers who work with radioactive materials or are exposed at work to man-made or natural radiation sources. The Committee has conducted a survey of countries world wide to obtain information that would allow comprehensive review of occupational radiation exposures.

158. Many workers in occupations involving exposure to radiation sources or radioactive material are individually monitored. One major exception is the large workforce exposed to enhanced levels of radiation from natural sources, for example, in parts of the extractive industries. The main reason for monitoring radiation exposures in the workplace is to provide a basis for controlling the exposures and for ensuring compliance with regulatory requirements and managerial policies. Both of these requirements go beyond the simple compliance with dose limits, and may include requirements to achieve and demonstrate the optimization of protection. Inevitably, the design and interpretation of monitoring programmes reflect local needs. There are advantages in extending these objectives to permit comparisons between different operations, if that can be done without too much difficulty. Such extensions would greatly assist the Committee in its compilations and comparisons of data.

159. For most workers involved with radiation sources or radioactive materials, the main sources of exposure are those external to the body. The doses due to internal sources are usually insignificant, apart from those due to the radon naturally present in all workplaces. Furthermore, it is much easier to monitor for external exposures than for internal ones. As a result, many workers are monitored for external exposures, even when their doses are expected to be low, but monitoring for internal exposure is carried out only when it is really needed. However, some areas of occupational exposure may not be adequately monitored. The extent and reporting of the occupational exposure in medical work is thought to be good in large medical installations, but it is likely to be less satisfactory in small installations.

160. It is not possible to make direct measurements of the effective dose to workers. In most monitoring for external exposure, the results from small personal monitoring devices are usually taken to be an adequate measure of the effective dose. The doses from internal sources are estimated from a number of measurements, including the amount of radioactive material excreted or retained in the body, and the concentration of radioactive substances in the air of the

workplace. The estimates depend on models of the time distribution of the intakes and of the transfer and retention processes in the body. Substantial uncertainties are inevitable.

161. There is some difficulty in presenting information about the typical individual dose to workers because policies for issuing monitoring devices differ. In particular, the widespread issue of monitoring devices to workers whose exposures are likely to be low artificially decreases the average recorded exposure of the exposed workforce. The Committee has made some use of the mean dose per measurably exposed worker, thus avoiding the distortion introduced by those who are monitored, but who receive trivial doses. Not all countries provide information in a form that allows this quantity to be estimated, so it cannot be used in the overall summary of data. For some purposes, the collective dose is a more satisfactory quantity, being little affected by the inclusion of large numbers of individually trivial doses.

162. There are wide variations between occupations in the recorded annual doses to monitored workers and also between countries for the same occupation. The detailed information from the Committee's review has allowed comparisons to be made between five-year periods from 1975 to 1989. This summary concentrates on the most recent quinquennium and comments on the trends over the previous periods. The world-wide average annual doses to monitored workers and the associated collective doses for 1985-1989 are summarized in table 3.

163. Workers in occupations involving adventitious exposure to natural sources, such as non-uranium mining, are not usually monitored and their doses are excluded from the figures in table 3. The principal occupations in this category are in aviation and mineral extraction industries. The annual effective dose to aircrew is typically between 2 and 3 mSv, with higher values in some supersonic aircraft. In the extractive industries, the annual effective doses are typically in the range 1-2 mSv in coal mines and 1-10 mSv in other mines. The annual occupational collective dose to these workers is estimated to be 8,600 man Sv. That estimate is quite uncertain, however, because of the limited monitoring data for those workers.

164. The estimates summarized in table 3 differ in some respects from those in earlier reports. These changes are due mainly to the improved database now available. The largest change is in the estimates of the doses from medical applications, much of which is due to radiation of low penetrating power. The personal dosimeters worn on the surface of the body then overestimate the effective dose, especially if, as is common, there is some partial shielding of the body by installed shields and protective aprons. The present estimate of collective dose is lower by a factor of 5 than the previous one and may still be too high by a factor of 2.

165. In the nuclear industry, the average annual collective dose has not varied substantially in the last 15 years, notwithstanding increases in electrical energy generated during that period by over a factor of 3 and in the number of workers by a factor of 2. The collective effective dose per unit of electrical energy generated declined by 50 per cent and the average individual dose by 30 per cent. Average individual doses are highest for workers in mining and milling operations. Reductions in individual doses to reactor workers come from a combination of improved operating practices and modifications to plants in the mid-1980s. Further improvements can be expected as new plants are commissioned.

166. There has been a decrease by a factor of about 2 in both individual and collective doses in general industry. Since the number of monitored workers has changed only slightly, this represents an overall improvement. In the defence

Table 3. Annual world-wide occupational exposures to monitored workers, 1985-1989

Occupational category	Annual collective effective dose a/ (man Sv)	Annual average effective dose per monitored worker (mSv)
Nuclear fuel cycle		
Mining	1 200	4.4
Milling	120	6.3
Enrichment	0.4	0.08
Fuel fabrication	22	0.8
Reactor operation	1 100	2.5
Reprocessing	36	3.0
Research	100	0.8
Total (rounded)	2 500	2.9
Other occupations		
Industrial applications	510	0.9
Defence activities	250	0.7
Medical applications	1 000	0.5
Total (rounded)	1 800	0.6
All occupations		
Grand total (rounded)	4 300	1.1

a/ Doses due to adventitious exposures to natural sources are not included. The annual collective dose from these natural sources is estimated to be about 8,600 man Sv, with the main contribution coming from underground, non-uranium mining. About half of that contribution comes from coal mining.

industries, both collective and individual doses have decreased, mainly owing to improvements in the operation and maintenance of nuclear-powered vessels.

167. When allowance is made for the overestimation in earlier reports, the occupational exposures in medicine show no trend in collective dose. There has been a reduction in the average individual dose, partly explained by an increase in the number of monitored workers.

168. It is rare for workers to be seriously exposed to radiation as a result of accidents. Minor incidents that cause unexpected, but not directly injurious, exposures are more frequent, but the policy for reporting them differs widely from place to place. The Committee has received information concerning about 100 accidents causing fatalities or having the potential to cause deterministic injuries in the workforce during the period since 1975. The list is almost certainly incomplete. The accident at Chernobyl was by far the most serious, causing 28 deaths from radiation-related causes. The doses to about 200 workers were high enough to cause clinical deterministic effects. Three deaths owing to radiation in other accidents have been reported. Accidents involving the public were discussed in section 5 above.

169. The collective dose due to exposures in minor accidents is included in the routine reports of occupational exposure. That due to serious accidents is not easy to estimate, but is certainly small compared with the total occupational collective doses. One component of collective dose that has not yet been

reported with other occupational exposures is that due to the emergency work undertaken to contain the damaged reactor at Chernobyl. This was not an accidental exposure, although it was the direct result of an accident. Some 247,000 workers were involved. The average dose from external exposure was estimated to be 0.12 Sv, giving a collective dose of about 30,000 man Sv. The doses from internal exposure varied during the work, but were mainly in the region of 10 per cent of those from external exposure.

7. Summary of current information

170. Typical collective effective doses committed by 50 years of practice for all the significant sources of exposure and by discrete events since the end of 1945 are shown in table 4. The bases for the values in this table are given in the earlier parts of this section, which, in turn, summarize the detailed evaluations given in the annexes to the present report.

171. Table 4 shows the relative importance of radiation sources in terms of the resulting collective doses. By far the largest source of exposure is the sum of natural sources. The whole world population is exposed to cosmic rays and radiation from naturally occurring radioisotopes of potassium, uranium, radium, radon, thorium etc. in soil, water, food and the body. The next most significant radiation source is the medical use of x-rays and radiopharmaceuticals in various diagnostic examinations and treatments. The doses from both diagnosis and treatment have been included in table 4, although they are not strictly comparable in terms of the resulting detriment.

Table 4. Collective dose committed to the world population by a 50-year period of operation for continuing practices or by single events from 1945 to 1992

Source	Basis of commitment	Collective effective dose (million man Sv)
Natural sources	Current rate for 50 years	650
Medical exposure	Current rate for 50 years	
Diagnosis		90
Treatment		75
Atmospheric nuclear weapons tests	Completed practice	30
Nuclear power	Total practice to date	0.4
	Current rate for 50 years	2
Severe accidents	Events to date	0.6
Occupational exposure	Current rate for 50 years	
Medical		0.05
Nuclear power		0.12
Industrial uses		0.03
Defence activities		0.01
Non-uranium mining		0.4
Total (all occupations)		0.6

172. Exposures from the atmospheric testing of nuclear weapons have diminished. There have been no further tests since the last one in 1980. Only small contributions to the collective dose are made by the generation of electrical energy by nuclear reactors, accidental events, and various occupational exposures, but these contributions are nevertheless important from the point of view of the radiation protection of individuals.

173. Apart from the doses from natural sources, the variation of individual doses over time and from place to place makes it impossible to summarize individual doses coherently. Some indications, however, can be provided.

174. The average annual effective dose from natural sources is 2.4 mSv, with elevated values commonly up to 10 or 20 mSv. Medical procedures in developed countries result in an annual effective dose to the average person between 1 and 2 mSv, of which about two thirds comes from diagnostic radiology. Average annual doses to individuals in the mid-1970s from atmospheric weapon tests were reported in the UNSCEAR 1977 report. By that time, most of the short-lived nuclides had decayed. The annual effective doses were about 5 μSv. Annual effective doses at the time of maximum testing were probably between 100 and 200 μSv in the northern hemisphere. Annual effective doses to the most highly exposed people near nuclear power installations are in the range 1-200 μSv. Occupational annual effective doses to monitored workers are commonly in the range 1-10 mSv.

V. THE PERCEPTION OF RADIATION RISKS

175. The word "risk" has several different meanings. It is often used descriptively to indicate the possibility of loss or danger, as in "the risks of hang-gliding". In technical contexts it is used quantitatively, but without any general agreement on its definition. Sometimes it is used to mean the probability of a defined adverse outcome, but it is also widely used as a combination of that probability and some measure of the severity of the outcome. These different meanings cause confusion among specialists but probably have little influence on the attitude of the general public. To the public, risk is largely descriptive or qualitative. Some risks are seen as worse than others partly because the outcome is thought to be more likely and partly because the outcome, if it occurs, is less welcome. There is little or no attempt to make a formal separation between these aspects or to combine them in anything more than an intuitive sense. Many factors influence the public's view of a risk. These include its source, its nature, the extent to which it is a familiar part of life, the degree of choice and control thought to be available to the individual, the confidence in the originator and regulator of the risk, and many others. Inevitably, any quantified discussion of risks involves both scientific and social judgements.

176. Against this background, there is no reason to expect the public attitude towards a risk to be the same as the attitude of those who estimate risks quantitatively, assess their importance and manage them. The task of the Committee is to provide quantitative estimates of the risk associated with ionizing radiation. The effects of exposure have been expressed in terms of the probability of their occurrence, the years of life lost in the case of fatal consequences and the severity of non-fatal consequences. The Committee is not concerned with making judgements about the relative importance of different kinds of risk to society or with the management of risks. It therefore aims to present its findings in a neutral way and has thought it desirable to take some account of the probable differences in the way its conclusions will be perceived by non-specialist readers.

177. The most important conclusion is that there is no uniformity of evaluation, comparison or acceptance of risks across individuals or societies. Considerable progress has been made, mainly during the last 20 years, in establishing a structured presentation of the factors that influence perceptions and in grouping them into classes. Some of the factors relate to the personal characteristics and experience of an individual, others are associated with the characteristics of the society in which the individual lives. Much depends on the individual's awareness of the source and character of the risks in question.

178. In all occupations and activities involving radiation, the quantification of and the perception of risks have been recognized as important issues. A major difficulty in managing risks has been to satisfy the concerns of individuals, communities and society. The basic approach in risk management has been to justify activities or practices by the benefits provided and to do all that is reasonable to reduce the risks. Views on the extent to which that approach has succeeded depend heavily on the perceptions of the viewer.

179. There are major difficulties in communicating information about radiation to the public. Even in countries that are highly developed technologically, many people do not know what radiation is, even in simple terms. Most of those who do know something about it associate it with accidents, weapons, fallout and cancer. Very few associate radiation with medical diagnosis or are aware of the normal background exposure to natural sources of radiation.

180. The Committee recognizes that many factors outside its remit influence the way in which its findings are viewed. Public concern about the levels and effects of radiation is more influenced by the perceived merits and social implications of the source of radiation than by the magnitude of the resulting exposures and risks. Nevertheless, the Committee recognizes its obligation to evaluate radiation exposures and to provide estimates of radiation risks that are soundly based, consistent and unbiased. The information must be trustworthy and clearly communicated if it is to contribute to achieving positive decisions for the whole of society.

VI. SUMMARY AND PERSPECTIVES

A. Levels of exposure

181. The Committee's estimates of the levels of exposure throughout the world are improving as the provision of data improves. As a very broad generalization, it can be concluded that improved procedures are decreasing the exposure per unit of practice by an amount that is sufficient to offset increases in the level of the practices.

182. Some sources of exposure continue at a constant level. Some continue over long periods, not necessarily at a constant level. Others are discrete events, or discrete series of events such as weapons tests. Sources that release radioactive materials to the environment deliver their doses over prolonged periods, so that the resulting annual doses do not provide a satisfactory measure of their total impact.

183. This report presents the collective dose to the world population received or committed from the end of 1945 to the end of 1992 (47 years) for discrete events and for a period of 50 years at the current rate of practice or exposure for all other sources. The results were shown in table 4.

B. Biological effects

184. The Committee's interest in the biological effects of radiation is mainly concentrated on the effects of low doses. Those effects have a low probability of occurring, but are serious when they do occur. Statistical limitations prevent epidemiological studies from providing direct estimates of risk at low doses, making it necessary to rely on radiobiology to provide a basis for interpreting the results of epidemiology. The combination of epidemiology and radiobiology, particularly at the molecular and cellular levels, is a useful tool for elucidating the consequences of low doses of radiation.

185. One of the most rapidly developing fields of work is concerned with the mechanisms of cancer induction as a result of changes in the molecular structure of DNA. Although rapid progress is also being made in the study of hereditary disorders, quantitative estimates of hereditary risk must still be derived from animal studies. Even the substantial exposures at Hiroshima and Nagasaki have not made it possible to obtain quantitative estimates of hereditary risks with a sufficient degree of confidence.

186. Despite the rapid progress in radiobiology and the increasing amount of data from epidemiology, the Committee has not yet found it necessary to make any substantial changes in its risk estimates.

C. Perspectives

187. The Committee's estimates of radiation exposure and its estimates of the risk of exposure indicate that radiation is a weak carcinogen. About 4 per cent of the deaths due to cancer can be attributed to ionizing radiation, most of which comes from natural sources that are not susceptible to control by man. Nevertheless, it is widely (but wrongly) believed that all the cancer deaths at Hiroshima and Nagasaki are the result of the atomic bombings. The studies in the two cities have included virtually all the heavily exposed individuals and have shown that, of 3,350 cancer deaths, only about 350 could be attributed to radiation exposure from the atomic bombings.

188. One way of providing a perspective on the implications of man-made radiation sources is to compare the resulting doses with those from natural sources. This is easy to do from a global point of view, which deals with total (or average) worldwide exposures. The collective doses were presented in table 4. However, many man-made sources expose only limited groups of people. The following paragraph attempts to distinguish between those situations.

189. On a global basis, one year of medical practice at the present rate is equivalent to about 90 days of exposure to natural sources, but individual doses from medical procedures vary from zero (for persons who were not examined or treated) to many thousands of times that received annually from natural sources (for patients undergoing radiotherapy). Most of the doses committed by one year of current operations of the nuclear fuel cycle are widely distributed and correspond to about one day of exposure to natural sources. Excluding severe accidents, the doses to the most highly exposed individuals do not exceed, and rarely approach, doses from natural sources. Occupational exposure, viewed globally, corresponds to about eight hours of exposure to natural sources. However, occupational exposure is confined to a small proportion of those who work. For this limited group, the exposures are similar to those from natural sources. For small subgroups, occupational exposures are about five times those from natural sources. The collective dose committed over 10,000 years by atmospheric nuclear testing is fairly uniformly distributed and corresponds to about 2.3 years' exposure to natural sources. That figure represents the whole programme of tests and is not comparable with the figures for a single year of practice. Only one accident in a civilian nuclear power installation, that at Chernobyl, has resulted in doses to members of the public greater than those resulting from the exposure in one year to natural sources. On a global basis, this accident corresponded to about 20 days' exposure to natural sources. Those findings are summarized in table 5.

Table 5. Exposures to man-made sources expressed as equivalent periods of exposure to natural sources

Source	Basis	Equivalent period of exposure to natural sources
Medical exposures	One year of practice at the current rate	90 days
Nuclear weapons tests	Completed practice	2.3 years
Nuclear power	Total practice to date	10 days
	One year of practice at the current rate	1 day
Severe accidents	Events to date	20 days
Occupational exposures	One year of practice at the current rate	8 hours

Notes

1/ The United Nations Scientific Committee on the Effects of Atomic Radiation was established by the General Assembly at its tenth session, in 1955 and its terms of reference were set out in resolution 913 (X) of 3 December 1955. The Committee was originally composed of the following Member States: Argentina, Australia, Belgium, Brazil, Canada, Czechoslovakia, Egypt, France, India, Japan, Mexico, Sweden, Union of Soviet Socialist Republics, United Kingdom of Great Britain and Northern Ireland and the United States of America. The membership was subsequently enlarged by the Assembly in its resolution 3154 C (XXVIII) of 14 December 1973 to include the Federal Republic of Germany, Indonesia, Peru, Poland and the Sudan. By resolution 41/62 B of 3 December 1986, the Assembly increased the membership of the Committee to a maximum of 21 members and invited China to become a member.

2/ For the previous substantive reports of UNSCEAR to the General Assembly, see Official Records of the General Assembly, Thirteenth Session, Supplement No. 17 (A/3838); ibid., Seventeenth Session, Supplement No. 16 (A/5216); ibid., Nineteenth Session, Supplement No. 14 (A/5814); ibid., Twenty-first Session, Supplement No. 14 (A/6314 and Corr.1); ibid., Twenty-fourth Session, Supplement No. 13 (A/7613 and Corr.1); ibid., Twenty-seventh Session, Supplement No. 25 (A/8725 and Corr.1); ibid., Thirty-second Session, Supplement No. 40 (A/32/40); ibid., Thirty-seventh Session, Supplement No. 45 (A/37/45); ibid., Forty-first Session, Supplement No. 16 (A/41/16); and ibid., Forty-third Session, Supplement No. 45 (A/43/45). These documents are referred to as the 1958, 1962, 1964, 1966, 1969, 1972, 1977, 1982, 1986 and 1988 reports, respectively. The 1972 report, with scientific annexes, was entitled Ionizing Radiation: Levels and Effects, Volume I: Levels, and Volume II: Effects (United Nations publication, Sales Nos. E.72.IX.17 and 18). The 1977 report, with annexes, was entitled Sources and Effects of Ionizing Radiation (United Nations publication, Sales No. E.77.IX.1). The 1982 report, with scientific annexes, was entitled Ionizing Radiation: Sources and Biological Effects (United Nations publication, Sales No. E.82.IX.8). The 1986 report, with scientific annexes, was entitled Genetic and Somatic Effects of Ionizing Radiation (United Nations publication, Sales No. E.86.IX.9). The 1988 report, with annexes, was entitled Sources, Effects and Risks of Ionizing Radiation (United Nations publication, Sales No. E.88.IX.7).

3/ To be issued as a sales publication.

APPENDIX I

Members of national delegations attending the thirty-eighth to forty-second sessions

ARGENTINA	D. Beninson (Representative), E. d'Amato, C. Arias, D. Cancio, A. Curti, E. Palacios
AUSTRALIA	K. H. Lokan (Representative)
BELGIUM	J. Maisin (Representative), R. Kirchmann, H. P. Leenhouts, P. H. M. Lohman, K. Sankaranarayanan, D. Smeesters
BRAZIL	E. Penna Franca (Representative), J. Landmann-Lipsztein
CANADA	E. G. Létourneau (Representative), A. Arsenault, D. R. Champ, R. M. Chatterjee, P. J. Duport, V. Elaguppilai, N. E. Gentner, B. C. Lentle, D. K. Myers
CHINA	Li Deping (Representative), Liu Hongxiang (Representative), Wei Lüxin (Representative), Leng Ruiping, Pan Zhiqiang, Tao Zufan, Wu Dechang
EGYPT	M. F. Ahmed (Representative), F. H. Hammad (Representative), F. Mohamed (Representative), H. M. Roushdy (Representative), S. E. Hashish
FRANCE	P. Pellerin (Representative), E. Cardis, R. Coulon, H. Dutrillaux, A. Flury-Hérard, H. Jammet, J. Lafuma, G. Lemaire, R. Masse
GERMANY a/	A. Kaul (Representative), W. Burkart, U. H. Ehling, W. Jacobi, A. M. Kellerer, F. E. Stieve, C. Streffer
INDIA	D. V. Gopinath (Representative), U. Madhvanath (Representative), N. K. Notani (Representative)
INDONESIA	S. Soekarno (Representative), S. Wiryosimin (Representative), K. Wiharto
JAPAN	H. Matsudaira (Representative), Y. Hosoda, T. Iwasaki, A. Kasai, S. Kumazawa, T. Matsuzaki, K. Nishizawa, H. Noguchi, K. Sato, K. Shinohara, S. Yano
MEXICO	E. Araico Salazar (Representative)
PERU	L. V. Pinillos Ashton (Representative)
POLAND	Z. Jaworowski (Representative), J. Jankowski, J. Liniecki, O. Rosiek, S. Sterlinski, I. Szumiel
RUSSIAN FEDERATION b/	L. A. Ilyin (Representative), R. Alexakhin, R. M. Barhoudarov, Y. Buldakov, V. Bebeshko, N. A. Dolgova, A. Guskowa, D. F. Khokhlova, Y. Kholina, E. Komarov, O. Pavlovski, G. N. Romanov

SLOVAKIA c/	M. Klímek (Representative)
SUDAN	O. I. Elamin (Representative), A. Hidayatalla (Representative)
SWEDEN	G. Bengtsson (Representative), L.-E. Holm, J. O. Snihs, L. Sjöberg
UNITED KINGDOM OF GREAT BRITAIN AND NORTHERN IRELAND	J. Dunster (Representative), R. H. Clarke, J. Denekamp, Sir Richard Doll
UNITED STATES OF AMERICA	F. A. Mettler (Representative), L. R. Anspaugh, J. D. Boice, C. W. Edington, J. H. Harley, N. H. Harley, C. Meinhold, P. B. Selby, W. K. Sinclair, E. W. Webster, H. O. Wyckoff

Notes

a/ At the thirty-eighth and thirty-ninth sessions: Federal Republic of Germany.

b/ At the thirty-eighth, thirty-ninth and fortieth sessions: Union of Soviet Socialist Republics.

c/ At the thirty-eighth, thirty-ninth, fortieth and forty-first sessions: Czechoslovakia.

APPENDIX II

Scientific staff and consultants cooperating with the Committee in the preparation of the present report

D. Beninson

B. G. Bennett

A. Bouville

R. Cox

J. Dunster

D. Goodhead

L. E. de Geer

J. Hall

L. E. Holm

G. N. Kelly

M. O'Riordan

W. J. Schull

P. Selby

J. W. Stather

J. Valentin

F. Vogel